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BUREAU of
RADIOLOGICAL HEALTH
Seminar Program

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# A PERSPECTIVE on POTENTIAL RADIATION HAZARDS

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# SELECTED PAPERS from the BUREAU of RADIOLOGICAL HEALTH SEMINAR PROGRAM

The Bureau of Radiological Health (BRH) Seminar Program is an outgrowth of several seminar program efforts conducted within the Bureau. The X-Ray Exposure Control Laboratory (XECL), the Radiation Bio-Effects Program, and the Research Grants Section had each established effective programs for the purpose of disseminating information. In 1968 these programs were combined into the Bureau of Radiological Health Seminar Program to encompass the total range of radiological health activities engaged in by the BRH.

The papers published in this series were originally oral presentations made at the XECL Seminar Program or at the BRH Seminar Program.

*The Selected Papers from the Bureau of Radiological Health Seminar Program* have been made possible through the cooperation of the guest speakers who have been dedicated to the advancement of the field of radiological health. They have given of their time and talents to prepare their oral presentations and these published manuscripts. In addition, the Seminar Program has been sustained through the continuous support of the entire professional staff of the Bureau of Radiological Health.

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Originally a native of Los Angeles, he attended the University of Southern California, receiving a B.A. degree magna cum laude in 1959. Following medical school, he returned to the West, interning at the King County Hospital, Seattle, Washington, from 1963-1964.

Having decided on a career in Radiology, he served a residency in radiology at the Columbia-Presbyterian Medical Center in New York City, from 1964-1967. While in residency, he became increasingly interested in therapeutic radiology and has authored several papers principally pertaining to malignant disease. He was certified by the American Board of Radiology in June of 1968.

Following two years service as a Radiologist with the Bureau of Radiological Health, he plans to take a two year fellowship in radiotherapy at the Stanford University Medical Center and plans to pursue a career in Academic Radiotherapy.

# A PERSPECTIVE ON POTENTIAL RADIATION HAZARDS [1]

J. R. Cassady, M.D.

## INTRODUCTION

The problem of real and potential hazards from ionizing radiation is one that constantly recurs and is the fundamental reason for the existence of the National Center for Radiological Health.[2] Recently, public attention has again been focused on these hazards to the extent that U.S. Congressional hearings were conducted.

In any discussion of potential radiation hazards, certain broad principals should be understood:

**X-radiation, except in relatively massive quantities, does not cause unique or pathognomonic effects. It merely increases the incidence or accelerates the appearance of already recognized conditions.**

**Statistical methods are necessary to prove virtually all low dose radiation effects. Therefore, experimental design, the handling of data, and the possibility of bias must be rigorously scrutinized.**

**Almost all postulated effects of x-radiation on humans in amounts less than approximately 100 R in a single exposure are effects extrapolated either from animal data or from much larger exposures to humans.**

Radiation physically interacts with tissue by ionization and excitation; without some form of interaction, no changes occur.

Unlike fire, certain chemicals, and many other agents potentially toxic to humans, x-rays are invisible, poorly understood by the majority, and potentially quite frightening to the general population. Therefore, it is incumbent that the possible hazards and effects of radiation be placed in perspective.

## SOMATIC EFFECTS

First, potential somatic hazards will be considered. By this is meant the effects that an individual exposed to ionizing radiation might experience within his lifetime. Three fundamental types of somatic damage are recognized. These can broadly be grouped as radiation-induced neoplasia, radiation damage to the whole body or a specific body organ or organ system, and finally, the broad topic of radiation-induced life shortening.

In considering each of these categories, considerable difference exists between high and low dose effects. Good data are available on the former and speculation by extrapolation on the latter.

Induction of neoplasia is one of the best known potentially harmful effects of x-ray or gamma radiation. Prior to discussion of specific examples of radiation induced tumors, brief mention should be made of possible mechanisms. Broadly, two principal mechanisms of neoplasia induction are postulated for radiation. They are:

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1. This paper was presented as a part of the X-Ray Exposure Control Laboratory's (Bureau of Radiological Health) Seminar Program on February 9, 1968, in Rockville, Maryland.
2. The National Center for Radiological Health became the Bureau of Radiological Health in January 1969 as a result of an administrative reorganization.

**Direct chromosomal effect**

**A) Mutation**
**B) Release**

**Indirect effect**

**A) Acceleration of inherent trait**
**B) "Favorable environment" theory**

A direct action on a cell or cells, presumably on the chromosomal material, which creates an abnormality such that in subsequent divisions that cell is uncontrolled by normal mechanisms, i.e., "malignant," and forms a "clone" or group of similar malignant cells.

Although incontrovertible evidence exists that radiation can create chromosomal abnormalities and subsequent *abnormal* divisions (i.e., genetic mutations), no good direct evidence exists to support the theory that mechanisms such as this underlie human malignancy. With one unsupported exception, no one has irradiated an otherwise untreated human cell (or cells) and seen that cell *become malignant* by subsequently transforming in tissue culture. (1)

It should be noted that a linear dose-response (where response is malignancy induction) relationship at any (even very low) dose level would exist with a mechanism whereby one single photon might strike the right target in a single cell and thus induce a malignancy.

The other postulated mechanism for radiation induction is indirect, whereby irradiation of an area would either create an acceptable environment for some other agent (i.e., a virus or chemical) to create a malignancy or would release from the cells irradiated a substance (i.e., virus) which, given proper conditions, might develop a tumor. Also, cells stimulated to regenerate an irradiated depopulated area might become malignant during this radiation-induced abnormal stimulation. Probably (although not necessarily) a threshold dose would be required and a rapidly rising slope of malignancy induction with progressively smaller increments of dose with an indirect mechanism such as this.

It is interesting that for this latter example, direct evidence is available in animal experimentations. One of the best examples are the murine leukemias studied primarily by Ludwik Gross and H. S. Kaplan. (2, 3)

In these studies, although radiation was required for leukemia development, the area or organ (primarily thymus) where the tumor subsequently developed did not necessarily have to be irradiated; i.e., an **indirect action.** This fact led to the discovery of the viral-like etiology of these tumors with radiation acting as a probable *releaser* of preexisting virus in cells, other than those where it would subsequently *settle* and induce a tumor.

In point of fact, no good evidence exists as to the mechanism of radiation induction of neoplasia in humans. It is obvious that this is a necessary piece of information in order to establish meaningful public health standards.

Principal studies in relating high doses to induction of neoplasia have emerged from analysis of such diverse exposures as:

**Radium dial painters with resultant bone sarcomas. (4)**

**Effects of thorium containing radiographic contrast agents (principal effects are hemangio-endothelioma of the liver and undifferentiated squamous cell carcinoma of the maxillary antrum). (5)**

**A variety of skin cancers and leukemia in early, overexposed radiation workers, i.e., Madame Curie. (6)**

**A high level of lung cancer in miners of pitchblende in Schneeberg in Austria and, possibly, of uranium in the Colorado plateau. (7, 8)**

**Leukemia in Japanese exposed to the A-bomb and British patients with ankylosing spondylitis treated by spine x-radiation. (9, 10)**

**Thyroid cancer in patients receiving face and chest irradiation in infancy or childhood, especially for acne and *status thymolymphaticus*. (11)**

**Bone tumors arising in patients receiving therapeutic x-radiation. (12)**

**An apparent increase in leukemia in patients treated with phosphorus-32 for polycythemia vera. (13)**

**Children treated with x-radiation for retinoblastoma. (14)**

Some generalizations gained in reviewing these and other pertinent reported series are:

The total dose received appears to have a direct effect on the percent incidence when doses of this magnitude are studied. However, a recent article on experimental tumor production in animals would tend to contradict this point. (15)

Although some carcinomas developed, the malignant tumors induced are frequently sarcomas, especially osteogenic sarcomas possibly related to the increased photon absorption in bone at low energy levels.

The magnitude of the dose has no apparent effect on shortening the *latent period* (i.e., the interval between the exposure to radiation and the first sign of tumor).

The tumors induced developed directly in the area irradiated and not outside the treatment volume.

In general, with the theoretical exception of leukemia, these observations are common to almost all reported radiation-induced human tumors.

Although the local effect is striking, whether this effect is direct or indirect (i.e., creation of an appropriate environment) is not known. The dose-response curves would, however, support a log dose-response mechanism with an exponentially rising slope.

In human adults, no convincing studies have thus far been reported relating a dose of less than 100 rads in a single exposure with any increased incidence of neoplasm induction.

While on the subject of radiation-induced malignancies, mention must be made of the only studies which would tend to show that more conventional diagnostic *low doses* of radiation may have harmful effects on specific populations. The studies primarily of Alice Stewart and Brian McMahon show, in a relatively convincing statistical manner, that children of mothers who received abdominal irradiation during pregnancy tend to have a higher incidence of leukemia and other malignancies than children who were not irradiated. (16, 17) Based on these reports, Court-Brown, Doll and Bradford Hill conducted an extensive prospective study in which they followed 39,166 live-born children whose mothers had received abdominal or pelvic irradiation during pregnancy (1945-1956). (18) Nine of an expected 10.5 were found to have died of leukemia before the end of 1958.

Based on the Hiroshima and Nagasaki data, as well as on Court-Brown and Doll's studies on ankylosing spondylitis, Lewis attempted an extrapolation of an assumed linear relationship down to the very small doses of natural background and estimated that a given percent of all cases of leukemia were related to this radiation. (19) This approach is open to considerable criticism which has principally been supplied by Austin Brues. (20) In

addition, studies attempting to relate natural background levels to leukemia incidence have been (to my knowledge) uniformly unsuccessful; however, here again, the statistical requirements of such studies are legion. (21) At this time the presence or absence of a true threshold is a matter for speculation.

In summary, with the possible exception of the fetus *in utero*, exposure to radiation levels achieved in present day diagnostic radiology carries a minute statistical risk (if, indeed, any risk at all) of neoplasia induction, such that a practical threshold can be said to exist for this phenomenon.

A substantial body of data has been gathered on whole body and individual organ effects of radiation. As excellent reference works are available on this material, let us only briefly review certain aspects here. (22) The best data for whole body effects of radiation on man come from the Japanese A-bomb explosions, atomic reactor accidents, and experience with patients with leukemia and polycythemia rubra vera (a blood disease) treated with whole body irradiation.

Generally, a whole body exposure of at least 50-100 R of relatively high energy radiation is required for routine development of symptoms. Below this level, a definite decrease in lymphocytes (a whole blood cell) occurs with exposures down to about 12 R (total body) or less, and chromosome abnormalities of unknown significance and/or importance have been reported, but little else has been documented.

Figure 1 illustrates some of the generally quoted radiation reactions to whole body radiation in varying doses. Four, and possibly five, syndromes occur following exposure to relatively large amounts of radiation (Fig. 2). Generally the quantity, quality, and type of radiation and, in addition, individual variation determine the type, severity, and time sequence of response.

| | |
|---|---|
| **Initial stage** | **0-40 hours** |
| **Latent stage** | **2-3 weeks** |
| **Manifest illness stage** | **3-6 weeks** |
| **Recovery stage** | **8-15 weeks** |

**DOSE RESPONSE**

**50 rads whole body - minimal findings**

**100 rads - most (89%) show no symptoms, 15 percent show mild acute radiation syndrome**

**200 rads - most show significant clinical and laboratory findings. A few fatalities have been reported.**

**450 rads - LD 50-30 with median therapy. Fatalities should be rare at less than 500 rads.**

**800-1000 rads - rather uniform fatalities**

**Fig. 1 Radiation reactions to whole body x-ray**

**Mild acute radiation syndrome - transient nausea and vomiting followed by laboratory and/or clinical signs of hematological damage**

**Hematologic**

**Gastrointestinal**

**Central nervous system**

**Vascular collapse (?)**

**Fig. 2 Radiation induced syndromes**

In terms of specific organ or organ syndromes system irradiation, the lower levels for demonstrable abnormalities are about the same. Thus, it generally takes a minimum of about 200 R in a single exposure to create a radiation cataract with a reported low dose of 50 R and an average gamma or x-ray photon dose of about 500-1000 R. (23) An exposure of lower magnitude will by no means result in uniform development of cataracts in those exposed. The *linear energy transfer* **(LET)** of the radiation used is quite important in this regard, with high LET particles, such as neutrons or alpha particles, being much more cataractogenic than gamma or x-ray photons.

Aside from the extraordinary sensitivity of circulating lymphocytes and a relative sensitivity of both male and female germinal cells at certain stages, organ doses below 50 R cumulative have produced no discernible effect. Doses of this magnitude cause observable effects in blood and gonads, but no proven late deleterious effects.

The problem of potential life shortening and aging effects of radiation might now be considered. It is easy to see how an agent which may cause a severe congenital anomaly, leukemia, or other malignancy could shorten the average lifespan in a group of sufficiently exposed individuals. These effects, however, are reasonably well quantitated for many exposure levels and types of radiation and, at the levels of radiation generally used in diagnostic radiology, with the possible exception of the fetus *in utero*, are not statistically significant.

In fact, a rather impressive body of evidence which shows that relatively low levels of radiation do not cause detectable life shortening exists. To enumerate, these studies would include an analysis of death rates among British radiologists, survivors of the Hiroshima and Nagasaki bombings, and more recently, present day American radiologists. (24, 25, 26) The frequently quoted early studies of Shields Warren on the longevity of American radiologists are not applicable for a number of reasons: (27)

> **A great number of these doctors practiced with either no or only a poor conception of the dangers of x-rays, especially as regards fluoroscopy. Therefore, rather than a *low-dose* group, these people represent a heterogeneous group, many of whom received very large doses.**
>
> **A suitable control group was not included. Early radiologists were representative of neither physicians as a whole, nor the general population, as many were crippled, had severe debilitating res-**

**piratory illnesses, etc; and not infrequently, these men entered radiology because it was one of the few remaining fields of medicine for which they could physically qualify.**

A much more difficult area to define and study is *aging* or the gradual deterioration of body processes that effects us all to a lesser or greater degree, finally culminating in death. Books have been written in an attempt to define just what aging is, but there has been no great agreement. The causes and mechanisms of aging are even more poorly understood. (28) Generally, however, radiation is implicated as a probable accelerating mechanism, and here the effect would be general rather than specific, as with a congenital anomaly or a malignancy. In this regard, the process might also accelerate the appearance of a malignancy or other specific diseases that otherwise would appear months or years later. Again, however, no pertinent, positive low-dose studies exist; only relatively high total body or large-field irradiation studies, generally of animals other than man which are then extrapolated to low doses on humans, exist.

In effect, it can be stated with some assurance that relatively high increments of daily, whole body radiation (generally greater than 10 R) over a prolonged period appear to shorten life significantly. (29) In addition, the early radiation workers, who were almost certainly exposed to daily doses (generally not whole body) of approximately the same order of magnitude, definitely suffered morbidity, and also, probably suffered life shortening. (6) However, it can also be stated with approximately equal assurance that present day radiation workers, who practice reasonable safety precautions, live as long, and probably die of the same diseases that they would have, had they not been radiation workers.

## GENETIC EFFECTS

As enumerated in an earlier Carmen Lecture of the RSNA, the postulated human genetic hazards from x-radiation, both diagnostic and therapeutic, formerly rested on five major propositions: (30)

**X-radiation causes gene mutations**

**The production of mutation by x-radiation is linear with dose and independent of dose rate**

**Most mutations are *harmful***

**The dose of ionizing radiation received from the natural background sources may contribute a substantial fraction of the *spontaneous* mutation rate**

**Most mutations are recessive**

One may add that there appears, from experimental data, to be a marked species variability between different types of organisms, probably related to DNA content, and possibly to varying recovery possibilities. It should again be noted that when radiation induces mutations with subsequent congenital anomalies and disease, they are the same pathologic entities that occur spontaneously, only with a higher incidence.

Since the Carmen Lecture (1958) previously noted, a great deal of new (and revolutionary) data has been discovered regarding radiation induced genetic effects.

Principally, they are:

In exposed (A-bomb) Japanese, a significant rise in abortions, still births, observable congenital anomalies, and life shortening was tested for and has not been observed. (25) An apparent change in the sex ratio of offspring

...as thought to be present initially; however, recent data tend to refute the validity of this finding.

Carter and colleagues irradiated male mice with 37.5 R (total body exposure) over a period of 5 weeks and studied the progeny. In 99,000 control offspring, six recessive and two dominant mutants were found; in 53,000 offspring or irradiated fathers, five recessive and one dominant mutation occurred. The difference is probably not significant. (31)

Male and female rats have been exposed in a continuous radiation field of 2, 5, or 10 R per day and have produced six successive litters without any abnormal offspring. One litter (the sixth) in the 10 R group, which was conceived after each parent had received about 3,300 R, was reduced in size of average individual. Young from the third litter of the 5 R group were mated; their parents had received 619 R, and the young rats received 282 R prior to mating. Normal litters resulted. (31) Although the data from both of the cited experiments can be criticized as not necessarily being representative of humans, barring the fervently hoped for absence of data from additional A- (or H) bombs, extrapolation seems to be the only likely alternative.

Studies by the Russells, and others, have shown that at low dose rates, genetic effects of x-radiation are not cumulative. (32, 33) That is, a considerable difference exists in the number of mutations caused by a single dose, as compared to that same dose fractionated over several increments in time. This latter finding appears to be related to the LET of the radiation and subsequent repair phenomena. This reduction is not readily observed with neutron irradiation.

Therefore, the original five propositions can be reexamined and the following statements can now be made with some assurance:

X-radiation can cause gene mutations. These are generally **recessive** and **harmful.**

The dose of ionizing radiation received from natural background sources may contribute a substantial fraction of the *spontaneous* mutation rate; however, this now appears unlikely. When more data on areas of high natural background become available, this point may become clarified.

The production of mutations by radiation is not linear with dose, and is not independent of dose rate; repair of genetic damage can and does occur.

## SUMMARY

This discussion has attempted to show where opinions on potential radiation hazards are based on fact, on extrapolation, or on pure speculation. It is obvious that the low dose effects of radiation postulated are almost entirely based on extrapolation and speculation. This presentation has also attempted to show that, assuming the extrapolations performed are valid (which they almost certainly are not), the magnitude of any potential hazard of medical x-ray to the general public under current radiologic practice is almost unobservable if, indeed, present at all. This does not mean, however, that safety precautions need not be observed. It merely emphasizes that, if they are utilized, and if, insofar as is practical, unnecessary x-radiation is eliminated, the public should have no fear of potential consequences of medical x-rays and, in fact, should constantly derive great benefit from them. In this regard, the typical *scare* headlines to which the general public are occasionally exposed are mentioned only for condemnation.

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Perspective on potential radiation hazards

U.S. Department of Health, Education and Welfare, Consumer Protection and Environmental Health Service, Environmental Control Administration, Bureau of Radiological Health (1969)

ABSTRACT: This discussion attempts to show where opinions on potential radiation hazards are based on fact, extrapolation, or pure speculation. It is obvious that the low dose effects of radiation postulated are almost entirely based on extrapolation and speculation. This discussion also attempts to show that, assuming the extrapolations performed are valid (which they most certainly are not), the magnitude of any potential hazard of medical x-ray to general public under current radiological practice is almost unobservable. The author emphasizes that this does not mean that safety precautions need not be observed but rather that, if they are utilized and unnecessary x-radiation is eliminated, the public should have no fear of potential consequences of medical x-rays.

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